
Regulation of Ca^{2+} signaling in pulmonary hypertension.

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Public Summary:

Understanding the cellular and molecular mechanisms involved in the development and progression of pulmonary hypertension (PH) remains imperative if we are to successfully improve the quality of life and life span of patients with the disease. A whole plethora of mechanisms are associated with the development and progression of PH. Such complexity makes it difficult to isolate one particular pathway to target clinically. Changes in intracellular free calcium concentration, the most common intracellular second messenger, can have significant impact in defining the pathogenic mechanisms leading to its development and persistence. Signaling pathways leading to the elevation of $[Ca^{2+}]_{cyt}$ contribute to pulmonary vasoconstriction, excessive proliferation of smooth muscle cells and ultimately pulmonary vascular remodeling. This current review serves to summarize some of the most recent advances in the regulation of calcium during pulmonary hypertension.

Scientific Abstract:

Understanding the cellular and molecular mechanisms involved in the development and progression of pulmonary hypertension (PH) remains imperative if we are to successfully improve the quality of life and life span of patients with the disease. A whole plethora of mechanisms are associated with the development and progression of PH. Such complexity makes it difficult to isolate one particular pathway to target clinically. Changes in intracellular free calcium concentration, the most common intracellular second messenger, can have significant impact in defining the pathogenic mechanisms leading to its development and persistence. Signaling pathways leading to the elevation of $[Ca^{2+}]_{cyt}$ contribute to pulmonary vasoconstriction, excessive proliferation of smooth muscle cells and ultimately pulmonary vascular remodeling. This current review serves to summarize some of the most recent advances in the regulation of calcium during pulmonary hypertension.

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